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Office of Administrative Law Judges
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Issue Date: 07 September 2005

CASE NO.: 2005-LHC-00577

OWCP NO.: 01-160187

In the Matter of

JANICE NEIN, Widow of
PHILLIP L. NEIN
Claimant

v.

BATH IRON WORKS CORPORATION
Employer

and

ONE BEACON INSURANCE COMPANY,
f/k/a COMMERCIAL UNION INSURANCE COMPANY
Carrier

Appearances:

G. William Higbee (McTeague, Higbee, Case, Cohen, Whitney & Toker), Topsham, Maine, for the Claimant

Richard van Antwerp (Robinson, Kreiger & McCallum), Portland, Maine for the Employer and Carrier

Before: Daniel F. Sutton
Administrative Law Judge

DECISION AND ORDER AWARDING BENEFITS

I. Statement of the Case

Janice Nein (the "Claimant") brings this claim against the Bath Iron Works Corporation ("BIW") for disability and death benefits under the under the Longshore and Harbor Workers' Compensation Act, as amended, 33 U.S.C. § 901, *et seq*. (the "LHWCA"), alleging that her late

husband, Phillip Nein (the "Decedent"), contracted fatal liver disease as a result of occupational exposure to carbon tetrachloride during the course of his employment at BIW as a shipyard electrician. The parties were unable to resolve the claims through informal proceedings before the Office of Workers' Compensation Programs ("OWCP"), and the OWCP transferred the case to the Office of Administrative Law Judges ("OALJ") for a formal hearing on the claim pursuant to section 19(d) of the LHWCA. 33 U.S.C. § 919(d).

Pursuant to notice, a hearing was conducted in Portland, Maine on May 23, 2005, at which time all interested parties were afforded an opportunity to present evidence and argument. The Claimant appeared at the hearing represented by counsel, and an appearance was made by counsel representing BIW and its insurance carrier, One Beacon Insurance Company, f/k/a Commercial Union Insurance Company ("One Beacon").[1] The Claimant testified at the hearing, and documentary evidence was admitted without objection as Claimant's Exhibits ("CX") 1-11 and One Beacon ("OBX") 1-5. Hearing Transcript ("TR") at 9, 11. The parties also offered written stipulations which were admitted as Joint Exhibit ("JX") 1. TR 8. One Beacon's request for leave to develop and introduce surrebuttal evidence outside of the time limits established in the pre-hearing order for the exchange of evidence prior to the opening of the hearing was taken under advisement and denied in a post-hearing order issued on May 27, 2005.[2] This order also set July 15, 2005 as the deadline for submission of post-hearing briefs. The Claimant timely filed a brief and a petition for attorney's fees. Although the brief on behalf of BIW and One Beacon was not filed until August 10, 2005, it has been considered in the absence of any objection. At the Claimant's request, issuance of this decision has been expedited due to economic hardship.

After careful analysis of the evidence contained in the record and the parties' arguments, I conclude that the Claimant has proved that the decedent's liver disease, disability and death arose out of and in the course of the Decedent's employment at BIW. Accordingly, I will award her disability and survivor's compensation, funeral expenses, interest, medical care and attorney's fees. My findings of fact and conclusions of law are set forth below.

II. Benefits Claimed

The Claimant seeks awards of (1) permanent partial disability compensation from August 23, 2003 through March 30, 2004 under section 8(c)(23) of the LHWCA, (2) funeral expenses and survivor's benefits commencing on March 31, 2004 under section 9 of the LHWCA, (3) medical expenses under section 7 and (4) attorney's fees under section 28.

[1] Prior to the hearing, unopposed motions to dismiss other parties, who had been identified in the informal proceedings below as potentially liable, were allowed, and a determination was made that One Beacon if the responsible party for any benefits awarded on the claim. *See* Administrative Law Judge Exhibit ("ALJX") 16.

[2] Specifically, BIW and One Beacon requested an opportunity to have their medical expert respond to testimony elicited from the Claimant's medical expert, Dr. Pohl, at a deposition that was taken with the agreement of all parties to afford the Claimant an opportunity to develop evidence in rebuttal to a report from the Respondents' medical expert that was served on the Claimant's attorney shortly before the deadline for exchange of evidence. This request was denied based on my finding that Dr. Pohl's deposition testimony constituted appropriate rebuttal and raised no new fact, analysis or medical theory warranting surrebuttal. ALJX 19.

III. Stipulations and Issues Presented

The parties offered the following stipulations: (1) the LHWCA applies to the claim; (2) the date of the alleged injury is August 23, 2003; (3) the Decedent died on March 30, 2004; (4) there was an employer-employee relationship at the time of the alleged injury; (5) the claim was timely filed and notices were timely given; (6) the informal conference was held on November 18, 2004; (7) the Decedent was a voluntary retiree, and the applicable National Average Weekly Wages for any benefits is $515.39; and (8) the Decedent's permanent partial disability was 100 percent during the period of August 28, 2003 through March 30, 2004. JX 1. The parties also agreed that the sole issue presented for adjudication is whether the Decedent's cirrhosis was causally related to his employment at BIW. *Id.* at 2; TR 8.

IV. Findings of Fact and Conclusions of Law

A. Background

The Decedent, who was born in 1934, was hired by BIW as an electrician on February 17, 1959. CX 5 at 15.[3] He was promoted to a position as a first class leadman in BIW's Electrical Department on July 22, 1968. *Id.* In February of 1973, he was transferred from the Electrical Department to the Estimating Department where he worked as a buyer until his retirement on March 1, 1995. *Id.* at 16, 18. He married the Claimant on November 29, 1955, and they remained married until the Decedent's death on March 30, 2004. CX 4 at 5, 12.

B. Evidence of Carbon Tetrachloride Exposure

The Claimant introduced the deposition testimony of two of the Decedent's former coworkers at BIW, Robert Westlake and Richard Rackliff, who described the use of carbon tetrachloride by electricians at BIW. Mr. Westlake testified that he worked in the BIW Electrical Department from 1961 to 1975 with the exception of a period from late 1969 to early 1973 when he left BIW to work in county government. CX 7 at 40-41. He said that the Decedent was his leadman during part of the time that he worked in the Electrical Department and that he worked with the Decedent on many occasions. *Id.* at 42. Mr. Westlake testified that he used carbon tetrachloride, both as an aerosol spray and as a liquid applied with a rag or brush, "for a multitude of things" including cleaning cables and electrical contacts and switches. *Id.* He said that he was sure that the Decedent also used carbon tetrachloride because they both worked in the same place. *Id.* at 42, 45. He said that workers in the Electrical Department used carbon tetrachloride on a daily basis during the early phases of ship construction when the electricians prepared cables for installation. *Id.* at 43. He also said that he and the Decedent used toluene in a large trough in which cables were submerged to remove waterproofing. *Id.* at 43-44. According to Mr. Westlake, the carbon tetrachloride and toluene both gave off noticeable odors. *Id.* at 42, 44. He recalled that carbon tetrachloride at BIW was replaced by something labeled "safety solvent" sometime prior to 1975 when he left the Electrical Department. *Id.* at 44-45. On cross-examination, he said that he believed that BIW discontinued the use of carbon

[3] Page citations to the Claimant's exhibits are to the "Bates stamp" numbers affixed to the bottom right corner of each exhibit rather than the original page numbers of each exhibit. One Beacon's exhibits are not "Bates stamped", so page citations to these exhibits will use the individual exhibit's page number.

tetrachloride before he left the Electrical Department in 1969. *Id.* at 46. He repeated that he personally saw the Decedent working with carbon tetrachloride and that he was certain that the Decedent used the substance. *Id.* at 47.

Mr. Rackliff testified that he worked at BIW for 40 years between 1950 and 1990 and that he spent 30 years between 1950 and 1980 in the electric shop. CX 6 at 22-23. He said that he used carbon tetrachloride as an electrician and knew that the Decedent would have used it as well for the same purposes. *Id.* at 24. He said that carbon tetrachloride was the only solvent that the electricians used to clean cables and equipment, but he also described a trough where "toluol" was used to strip cables. *Id.* at 25-27. He further testified that no electricians wore any breathing protection or gloves during the 1960s and that he believed that the Decedent would have inhaled fumes while using chemicals in the Electrical Department. *Id.* at 27, 29. On cross-examination, Mr. Ratliff testified that he only used "toluol" because he was a laborer and was not allowed to touch electrical components, but the Decedent worked as an electronic technician and used carbon tetrachloride for as much as five or six hours per day to clean electronic equipment and cables. *Id.* at 30. He also stated that he probably never worked with the Decedent "side by side" and could not testify that he ever saw the Decedent actually work with either "toluol" or carbon tetrachloride. *Id.* at 32, 36.

BIW and One Beacon introduced the deposition testimony of Charles Crosby, Jr. who worked at BIW from 1954 until 1995, primarily as a supervisor in the Electrical Department. OBX 4 at 3. Mr. Crosby testified that he did not work directly with the Decedent as they were involved in different phases of ship construction – he worked with combat systems while the Decedent worked in electronics calibration. *Id.* at 6. He said that carbon tetrachloride was available in the Electrical Department through the machine shop tool crib between 1959 and 1968. *Id.* at 7. He added that carbon tetrachloride was a good degreasing agent that was not considered hazardous in those years. *Id.* at 7-8. He was asked about the uses of carbon tetrachloride and responded that he felt that it would have been used to degrease older switches and contacts during ship overhauls but not in new ship construction. *Id.* at 8-9. He further testified that BIW did not do any overhaul work from 1959 until 1967 when the first overhaul work began. *Id.* at 9. However, he added that BIW did its own calibration work so that "it's possible some occasions might have occurred where something was used, carbon tet was used on something there." *Id.* at 10-11. Mr. Crosby said that had oversight responsibility for the Decedent's work after he became a leadman on July 22, 1968, and he said that the Decedent would not have been allowed under the terms of the union contract to "get his hands into the equipment" or "handle . . . the material himself" after he became a leadsman. *Id.* at 11-12. He did not recall the Decedent missing time from work due to illness or appearing to be sick when he worked in the Electrical Department. *Id.* at 17-18.

Of these three witnesses, Mr. Westlake is the only one who worked directly with the Decedent. Based on his testimony that he personally observed the Decedent working with carbon tetrachloride while he was employed in BIW's Electrical Department during the 1960s, I find that the weight of credible evidence establishes that the Decedent was exposed to carbon tetrachloride during the course of his employment at BIW. In this regard, it is noted that Mr. Crosby testified that he considers Mr. Westlake to be an honest and reliable witness. OBX 4 at 19. While Mr. Crosby testified that he did not believe that the Decedent would have used carbon

tetrachloride, he conceded that it was widely available at BIW during the 1960s when it was not considered to be dangerous and that it could have been used in the calibration lab where the Decedent worked. Moreover, he worked in different phases of the construction process and thus was not in as good a position as Mr. Westlake to observe whether the Decedent actually used carbon tetrachloride or any other chemicals in his work. Finally, Mr. Westlake's testimony that the Decedent worked with carbon tetrachloride at BIW is consistent with the history that the Decedent provided to his treating physician. *See* CX 10 at 116.

C. Medical Evidence

Seven years after his retirement from BIW, the Decedent developed swelling in his lower extremities and abdomen, and he was referred by his primary care physician to Richard P. Beveridge, M.D., a gastroenterologist, for evaluation. He was initially seen by Dr. Beveridge on July 15, 2002 when he complained of progressive peripheral edema in the lower extremities, increase in abdominal girth and 30-pound weight gain over the previous several months with a medical history of diabetes, irregular heartbeat, and hypertension. CX 10 at 60. He also reported to Dr. Beveridge that he had worked with carbon tetrachloride for two days, eight hours a day, at age 20 and developed "yellow eyes." *Id.* at 61.[4] Dr. Beveridge commented that the Decedent's work with carbon tetrachloride "for a few days in his 20's" was interesting. *Id.* at 62. The following year, the Decedent was admitted to the Mid Coast Hospital on August 26, 2003 at the request of Dr. Beveridge with a new complaint of right leg cellulitis. *Id.* at 75. He was evaluated by Carl DeMars, M.D. whose primary impression was "Cirrhosis, thought to be cryptogenic." *Id.* He was readmitted to the hospital on October 26, 2003, again with a complaint of pain and swelling in his right leg. *Id.* at 94. At this time, the admitting physician, Nancy Hasenfus, M.D., described the Decedent as a "retired BIW worker was also a train engineer for a number of years", and she wrote that "[h]e was exposed to carbon tetrachloride when he was in his 20's . . . [h]e used to [c]lean engines with it." *Id* [5] In a report of consultation dated December 8, 2003, Dr. Beveridge wrote,

> He did have extensive exposure to carbon tetrachloride while working at Bath Iron Works when he was in his 20s. He does recall having yellow jaundice at that point, but no further evaluation was carried out.

Id. at 116. Unfortunately, the Decedent's liver disease rapidly progressed, and on February 25, 2004, Dr. DeMars reported that he had "end stage cryptogenic cirrhosis with increasing anasarca and back pain." *Id.* at 130. Liver transplantation was explored but not pursued in light of the Decedent's age and other medical complications. *Id.* at 145, 162, 168. On March 8, 2004, he was seen by Peter Bridgman, M.D. who noted that the Decedent "has a history of cryptogenic

[4] Dr. Beveridge's notes do not indicate whether the Decedent described where his exposure to carbon tetrachloride occurred, but the Claimant was 20 years old in 1954, five years before he was hired by BIW. In this regard, it is noted that the Decedent's BIW employment records show prior employment between August of 1954 and February of 1959 as telephone installer and repairman, electrical foreman, maintenance man, "splicer" and electronic technician. CX 5 at 16.

[5] There is no other reference in the record to the Decedent ever having worked as a train engineer, so the source of this erroneous information about the Decedent's employment history is unclear. However, once part of the medical record, the error was repeated in later reports from Drs. Demars and Beveridge. *See* CX 10 at 113, 117, 128, 130.

cirrhosis, with an old history of carbon tetrachloride exposure though again it's not clear from Dr. Beveridge's note whether this was an obvious cause." *Id.* at 146. On March 19, 2004, Dr. Beveridge wrote that the Decedent had end stage liver disease "likely related to cryptogenic cirrhosis" which was "possibly related to carbon tetrachloride." *Id.* at 162-163. On March 22, 2004, he was transferred from the Mid Coast Hospital to the Maine Medical Center by Dr. Demars whose primary discharge diagnosis was cryptogenic cirrhosis "possibly related to carbon tetrachloride versus non-alcoholic steatohepatitis." *Id.* at 165.

On March 22, 2004, the Decedent was admitted to the Maine Medical Center with a principal diagnosis of end-stage liver disease with hepatorenal syndrome. CX 10 at 64. Two days later, he was discharged to hospice care with a poor prognosis. *Id.* at 65. The Decedent passed away on March 30, 2004, and Dr. DeMars certified the cause of death on the death certificate as cryptogenic cirrhosis. CX 2 at 5.

An autopsy was performed by Sujata Mukhopadhyay, M.D. who rendered the following final diagnoses: (1) macro and micronodular cirrhosis of the liver; (2) atherosclerotic cardiovascular disease; (3) thick fibrous plaques on the pleural and peritoneal surfaces of the diaphragm; (4) fibrous thickening of the pericardium and pericardial adhesions; (5) chronic bronchitis; and (6) patchy bronchopneumonia. CX 10 at 171. Dr. Mukhopadhyay concluded that the cause of death was progressive hepatic failure and encephalopathy. *Id.* at 172. She noted that the Decedent had a "past history of carbon tetrachloride exposure and cirrhosis of the liver possibly related to it." *Id.* at 172. She then made the following comments regarding the cause(s) of the Decedent's cirrhosis:

> The liver shows extensive changes of cirrhosis. According to the history there is evidence of carbon tetrachloride exposure. However, there is significant and chronic steatohepatitis ongoing in the liver. Definitive distinction between non-alcoholic steatohepatitis versus carbon tetrachloride associated hepatonecrosis and cirrhosis cannot be made at this stage of the disease."

Id. at 172. A second pathologist, Douglas A. Pohl, M.D., Ph.D. who was retained by the Claimant, reviewed the medical records and the autopsy report. Dr. Pohl earned his Ph.D. in pathology and is board-certified in anatomic and clinical pathology with a subspecialty in cytopathology. CX 11 at 196. He stated in his report that carbon tetrachloride is a well known hepatic and renal toxin and that "repeated exposure to carbon tetrachloride may produce progressive disease in the liver and kidney and ultimately leading to cirrhosis and heptorenal failure." CX 10 at 192. He noted that the Decedent had been extensively worked up for other causes of cirrhosis with negative findings, and he concluded that "in the face of Mr. Nein's significant exposure to a well known hepatotoxic agent (carbon tetrachloride), and the absence of any other causes, it is clear that Mr. Nein's cirrhosis was caused by his prior occupational exposure to carbon tetrachloride." *Id.* Dr. Pohl stated that pathologically, the early stages of carbon tetrachloride-induced hepatotoxicity are characterized by centrilobular necrosis and steatosis and that the progressive organ damage resulting from carbon tetrachloride exposure may be accompanied by cirrhosis or scarring of the liver with a slow or rapid progression to end stage hepatic failure. *Id.* He added that the Decedent's "clinical course is completely compatible with heptorenal failure arising as a consequence of carbon tetrachloride poisoning." *Id.* Finally,

Dr. Pohl stated that alcohol use is a known aggravating factor in patients with carbon tetrachloride exposure, but he noted that the medical records do not indicate that the Decedent was an alcoholic. *Id.*[6]

In response to Dr. Pohl's report, BIW and One Beacon introduced a report from Raymond D. Harbison, MS, Ph.D., a board-certified toxicologist and the Director of the Center for Environmental/Occupational Risk Analysis & Management in the College of Public Health at the University of South Florida ("USF"). EX 1. Dr. Harbison is a Professor of Environmental and Occupational Health at USF's College of Public Health, a Professor of Pharmacology and Pathology at USF's College of Medicine, and an Adjunct Professor of Medicine in the College of Medicine at the State Universtity of New York at Buffalo. *Id.* at 1. He earned his Ph.D. in toxicology in 1969, and he has published over 100 articles in the fields of toxicology and pharmacology. EX 2. Dr. Harbison reviewed the Decedent's medical records, the deposition testimony of the Decedent's coworkers and Dr. Pohl's report. EX 1 at 1. Regarding the Decedent's work history and exposure to carbon tetrachloride, Dr. Harbison stated that electricians are not known to have "excessive" carbon tetrachloride exposure, and he stated that the Decedent would not have been exposed to carbon tetrachloride after he became a leadman in 1968. *Id.* at 2. Regarding Dr. Pohl's conclusions, Dr. Harbison stated that any conclusion that the decedent's cirrhosis was causally related to carbon tetrachloride exposure would have to be based on reliable evidence of the following: (1) that there was a harmful amount of carbon tetrachloride in the workplace, (2) that the Decedent was exposed to a harmful amount of carbon tetrachloride, (3) that the dose and duration of his exposure was sufficient to cause end stage liver disease, and (4) that the Decedent's specific injuries resulted from exposure to carbon tetrachloride. *Id.* at 2. He then offered the following critique of Dr. Pohl's conclusions:

> Dr. Pohl has neither provided nor relied upon scientifically reliable evidence that Mr. Nein received a harmful carbon tetrachloride exposure at his workplace located at Bath Iron Works. Instead, he relies on the belief that any level of exposure to carbon tetrachloride could cause and did cause the liver condition of Mr. Nein. Dr. Pohl has not provided any evidence about or referred to any evidence of the level, duration, and frequency of exposure of Mr. Nein to carbon tetrachloride at his workplace. Dr. Pohl has provided no evidence that the level of exposure at Mr. Nein's workplace ever exceeded any level of carbon tetrachloride known to cause the liver condition of Mr. Phillip Nein. Dr. Pohl has no knowledge of the air levels of carbon tetrachloride in the workplace of Mr. Nein. Dr. Pohl has no knowledge of the actual release of carbon tetrachloride or the amount used or the concentration of carbon tetrachloride in the workplace of Mr. Nein. Instead he uses circular reasoning. He concludes that because Mr. Nein has end stage liver disease, he was exposed to sufficient carbon tetrachloride to cause the liver disease. This conclusion is not based on any scientifically accepted or reliable methodology. No reasonable practitioner, trained in science and medicine, would conclude that sufficient evidence exists for linking any unknown carbon tetrachloride exposure more than three decades earlier with the liver

[6] This appears to be an understatement as the medical records consistently describe the Decedent's consumption of alcohol as minimal. *See* CX 1O at 61, 76, 80, 94, 98, 103. 113, 116, 128, 130, 143.

conditions of Mr. Nein thirty years after discontinued exposure based on the facts of this case.

Id. at 2-3. Dr. Harbison further charged that Dr. Pohl has presented a "new, unique, and novel hypothesis . . . that an unknown level of carbon tetrachloride exposure that produced no clinical manifestations of liver injury at the time of exposure caused liver injury more than thirty years after discontinued use by Mr. Nein." *Id.* at 3. He further asserted that "[t]here is no biologically plausible mechanism that could lead to liver disease as a result of exposure to carbon tetrachloride that was discontinued more than thirty years earlier" and that Dr. Pohl's "extrapolation of an exposure to carbon tetrachloride that did not result in clinical manifestations thirty years earlier as a cause of liver disease thirty years after discontinued exposure to carbon tetrachloride is not consistent with toxicological principles and published literature." *Id.* Dr. Harbison also stated: (1) that Dr. Pohl did not cite any evidence that workplace carbon tetrachloride exposure at BIW exceeded the permissible exposure limit established by the Occupational Safety and Health Administration ("OSHA"); (2) that it is his understanding that the Decedent had no clinical manifestations contemporaneous with his use of carbon tetrachloride, and Dr. Pohl has not provided any explanation for the lack of clinical manifestations during the 1960s; (3) Dr. Pohl did not rule out other causes of the Decedent's liver disease; (4) Dr. Pohl's methodology relies on an "extrapolation" of exposure history provided by others, and his methodology does not require evidence of exposure levels sufficient to cause liver disease; (5) Dr. Pohl's analysis fails to comply with the principles and methods of toxicology; (6) there is no objective evidence that the Decedent was exposed to harmful or toxic levels of carbon tetrachloride at BIW; (7) Dr. Pohl did not provide any epidemiological literature of a "specific carbon tetrachloride-induced toxic effect concordant with that observed" in the decedent; and (8) Dr. Pohl's causation opinion is not "biologically possible" because "[c]arbon tetrachloride is rapidly eliminated from the body and could not cause Mr. Nein's liver injury in 2002." *Id.* at 4-7. Thus, Dr. Harbison concluded "to a reasonable degree of toxicological certainty . . . that workplace exposure to carbon tetrachloride at the Bath Iron Works' facility could not have caused the end stage liver disease and death of Mr. Nein." *Id.* at 7.

As mentioned above, Dr. Pohl was provided with an opportunity to respond to Dr. Harbison's report at a deposition taken on April 28, 2005. CX 11. He stated that he has performed autopsies on patients with carbon tetrachloride exposure and found that the primary organs affected were the liver and kidneys. *Id.* at 198. Dr. Pohl testified that he reviewed the Decedent's medical records as well as the deposition testimony from Messrs. Westlake, Rackliff and Crosby and that it was his conclusion that "in fact the carbon tetrachloride was the specific cause of [the Decedent's] cirrhosis and subsequent death." *Id.* at 199-200. He stated that he agreed with Dr. Mukhopadhyay's findings, except that he characterized the relationship between the Decedent's cirrhosis and carbon tetrachloride exposure as "probable" rather than possible. *Id.* at 201. Dr. Pohl explained that the toxic effect of carbon tetrachloride exposure has been studied in both humans and animals, and he said that carbon tetrachloride has been cited in medical schools for years as an example of a toxic agent that causes a particular type of liver damage known as centrilobular necrosis. *Id.* at 205. He testified that the Rabe study showed that carbon tetrachloride exposure over periods as small as five years is capable of causing liver damage, and he noted that the National Institute for Occupational Safety and Health ("NIOSH") published a revised recommended standard for carbon tetrachloride in 1977, stating that medical

records for persons occupationally exposed to carbon tetrachloride should be maintained for 30 years after termination of employment, "because the effects of carbon tetrachloride poisoning may not become apparent until many decades after the exposure occurred . . . ." *Id.* at 205-208.[7] In regard to Dr. Harbison's report, Dr. Pohl suggested that Dr. Harbison, as a toxicologist, does not have a pathologist's training and expertise to determine whether chemical exposure caused an injury or disease:

> The Ph.D. toxicologist such as Dr. Harbison looks at the chemicals and what their intended use is and the potential levels of those chemicals in various workplaces, but a Ph.D. toxicologist does not have an understanding of the pathology related to the damage that the chemicals actually caused, and in fact, a Ph.D. toxicologist would never examine slides from such a patient or be aware of the actual cellular changes that occur after someone has been exposed to a toxic substance. So his part of toxicology is limited to the non-medical part of the assessment of a substance and how much of a substance was present and what the likely damage that it could cause to someone might be, but he doesn't understand what the mechanisms are and what it looks like.

Id. at 210-211. Dr. Pohl also responded to Dr. Harbison's specific criticisms. He stated that the NIOSH publications and the epidemiological studies provided scientific evidence of a causal connection between carbon tetrachloride exposure and liver damage. *Id.* at 212. He testified that the fact that the Decedent's coworkers could detect the odor of carbon tetrachloride when they used it enabled him to determine that there was a harmful level of carbon tetrachloride in the Decedent's BIW workplace because humans can only detect carbon tetrachloride in concentrations of approximately ten parts per million while the current permissible exposure limit is established at .03 parts per million. *Id.* at 212-213. Thus, he testified that the best evidence of whether the Decedent and his coworkers at BIW were exposed to harmful levels of carbon tetrachloride at BIW is the fact that they were able to smell the substance. *Id.* at 213, 243. He said that the autopsy showed the presence of end stage cirrhosis which is a consequence of harmful carbon tetrachloride exposure, and regarding the level and duration of the Decedent's exposure, he stated,

> I think that it's clear from the coworker depositions as well as the medical records that he was exposed over a period of at least five or six years, perhaps slightly longer, that the exposure occurred on a regular daily basis, that the exposure was at a level known to be toxic to the liver, that is a level in which you can smell carbon tetrachloride in the air and therefore the level, duration and frequency of Mr. Nein's exposure was sufficient to produce cirrhosis as reflected in published medical articles.

Id. at 214. Regarding Dr. Harbison's comments about the absence of evidence that the Decedent had clinical manifestations of carbon tetrachloride-induced liver damage contemporaneous with his exposure, Dr. Pohl explained that like alcohol, carbon tetrachloride exposure can produce

[7] BIW's objection that Dr. Pohl's testimony regarding the NIOSH revised recommended standard lacked adequate foundation (CX 11 at 207) is overruled as I find that Dr. Pohl's expertise sufficiently qualifies him to render an opinion regarding the purpose of the medical records retention requirement.

damage to the liver without accompanying clinical symptoms and that the aggregate damage over a period of years results in cirrhosis which he described as an attempt by the liver to heal the damage caused by a toxic substance. *Id.* at 215. He was referred to the Cecil Textbook of Medicine, 22nd Ed. (2004) (*see* Deposition Exhibit 7, CX 11 at 422), which he identified as a reliable authority widely used by internists and primary care physicians, and he testified that the text's statement that 40 percent of cirrhosis patients are asymptomatic contradicts Dr. Harbison's point that the absence of evidence of clinical manifestations is significant. *Id.* at 216-217. He also pointed out that the phenomena of asymptomatic patients with cirrhosis is frequently seen in the alcoholic population. *Id.* at 244. Dr. Pohl further testified under cross-examination that the Decedent's medical records did show that he had contemporaneous clinical manifestations of carbon tetrachloride exposure:

> What I testified to was that at the time he used the carbon tetrachloride he sustained the damage to his liver. I think we see evidence of that from the record that you reviewed with me, the fact that he described yellow eyes which is jaundice. For someone to have yellow eyes, they have to have substantial amount of liver damage leading to systemic jaundice and very high bilirubin levels and if the worker, Mr. Nein, sustained that degree of jaundice that would indicate that he was at very high risk for developing cirrhosis as part of the reparative process on that jaundice.

Id. at 246.[8] Dr. Pohl disagreed with Dr. Harbison's assertion that he had not ruled out other possible causes of the Decedent's cirrhosis, stating that he had intensively searched the Decedent's records for potential causes of liver disease, such as hepatitis, toxic chemical exposures and chronic alcohol use, and that he was able to find only one causative agent -- carbon tetrachloride exposure. *Id.* at 219. He explained that damage to the liver occurs when cytochrome enzymes attempt to break down carbon tetrachloride that has been deposited in liver cells by the bloodstream:

> [T]hat process of changing the carbon tetrachloride actually causes damage to the liver cell, and if it's sufficient, it causes necrosis, which means death of the cell. If it is not sufficient enough to cause death, it can produce steatosis, which means that the liver cell is deranged and takes up too much fat, and if you have repeated exposures of the cell over a period of time on a variable basis, the altering episodes of cell death and repair, cell death and repair lead to fibrosis, which at its end is what we call liver cirrhosis.

[8] Dr. Pohl was questioned at length about the references in the medical records to the Decedent's "yellow eyes" reaction to carbon tetrachloride exposure and whether this incident occurred prior to the commencement of his BIW employment. CX 11 at 229-231. Dr. Pohl testified that he had not seen anything in the records to indicate that the Decedent had been exposed to carbon tetrachloride prior to his employment at BIW and that he was not aware of anything in the record that showed that the "yellow eyes" incident predated the BIW employment. *Id.* at 247. However, the Decedent's work history, as previously discussed, included several years of electrical and telephone work prior to 1959 when he was hired by BIW. While Dr. Beveridge in one report specifically placed the jaundice episode during the period of the Decedent's BIW employment (CX 10 at 116) , I find that there is simply too much ambiguity in the evidence to conclude that it is anything but inconclusive as to whether the "yellow eyes" incident occurred before or during the Decedent's BIW employment.

Id. at 224. Dr. Pohl agreed with Dr. Harbison's statement that any carbon tetrachloride that the Decedent absorbed during the 1960s would have been evacuated long before the diagnosis of cirrhosis was made in 2002, but he added that the damage to the liver caused by carbon tetrachloride remains and can become clinically manifest as cirrhosis years after exposure. *Id.* at 225-226. On cross-examination, he further explained that although the damage to the liver from carbon tetrachloride exposure occurs immediately, the damage does not always produce immediate symptoms because the liver has very few nerve fibers and typically does not exhibit symptoms that are contemporaneous with injury. *Id.* at 239. In this regard, Dr. Pohl testified that effect of carbon tetrachloride on the liver is similar to that produced by alcohol:

> If you take a college kid that goes out and drinks a lot the night before, when he wakes up the next morning he's got a miserable headache, he's probably suffered substantial damage to his liver, but his symptoms have nothing to do with the liver. They're mostly related to imbalances in fluid in his body producing his headache.
>
> Q. And if he stops drinking, then the liver will restore itself, isn't that right?
>
> A. It typically regenerates itself, but with alcohol use or carbon tetrachloride use that's chronic, it's the repeated insults to the liver that result in the permanent damage that ultimately lead to cirrhosis.
>
> Q. And it's your testimony today that it can take upwards of 30 or 32 or 33 years before the damage will manifest itself as cirrhosis, 30 to 32 years after the last interaction with that substance?
>
> A. Yes, one has to understand the cirrhotic process. Even in recovering alcoholics who have ceased drinking but developed cirrhosis at the end of their period of drinking, the cirrhosis can self-perpetuate itself, and this is related to what's called the tumor necrosis factor B agent which causes damage to the liver, even though the toxin is no longer there, leading to additional cirrhosis.

Id. at 239-240. He was then questioned about medical literature which notes that in most instances of carbon tetrachloride poisoning, the patient will develop signs of liver injury within a few days of exposure, and he responded that the referenced articles involve cases of "acute poisoning" which "produces massive amounts of damage to multiple different organs . . . and of course with that kind of damage that will in fact produce clinical symptoms." *Id.* at 241-242. He explained that these cases are distinguishable from the Decedent's exposure which he characterized as chronic low or intermediate level intoxication. *Id.* at 242. Again, he analogized carbon tetrachloride exposure to alcohol consumption:

> [U]sing alcohol as an example, it's the difference between drinking an entire liter of vodka versus just having a couple of shots. Obviously, a couple of shots isn't going to produce any clinical sequelae other than intoxication, but a whole liter is enough to put somebody in the hospital and on a respirator.

Id. at 242-243. Dr. Pohl was also questioned about Dr. Mukhopadhyay's statement that she was unable to make a definitive distinction between non-alcoholic steatohepatitis versus carbon tetrachloride associated hepatonecrosis and cirrhosis in the Decedent's case. He stated that Dr. Mukhopadhyay could not determine the cause of the Decedent's cirrhosis "pathologically because she did not have access to the other clinical materials that I did." *Id.* at 245. He explained that a pathologist often may not be able to determine the underlying cause of disease from examining a liver specimen, but must instead look to the patient's clinical and other histories in order to render a final assessment. *Id.* Lastly, Dr. Pohl testified that cases of either acute or chronic carbon tetrachloride poisoning are rare, that he has only performed three or four pathological examinations in carbon tetrachloride cases and that the Decedent was the first case of alleged carbon tetrachloride exposure at BIW that he had seen. *Id.* at 251-254.

D. Causation

The sole issue presented is whether the Decedent's exposure to carbon tetrachloride during the course of his employment at BIW caused his liver cirrhosis and, ultimately, his disability and death. To prove this causal connection and thereby establish that the Decedent's cirrhosis is covered by the LHWCA, the Claimant bears the initial burden of making out a *prima facie* case. That is, she "must at least allege an injury that arose in the course of employment as well as out of employment" and show that the Decedent "sustained physical harm and that conditions existed at work which could have caused the harm." *Bath Iron Works Corp. v. Brown*, 194 F.3d 1, 4 (1st Cir. 1999) (*Brown*), quoting *U.S. Indus./Fed. Sheet Metal, Inc. v. Director, OWCP*, 455 U.S. 608, 615 (1982) and *Susoeff v. San Francisco Stevedoring Co*., 19 BRBS 149, 151 (1986). "[T]he claimant is not required to show a causal connection between the harm and his working conditions, but rather must show only that the harm could have been caused by his working conditions." *Bath Iron Works Corporation v. Preston*, 380 F.3d 597, 605 (1st Cir. 2004) (*Preston*). BIW and One Beacon contend that the Claimant has not carried her *prima facie* burden. Respondents' Brief at 4-5. The Claimant has alleged that the Decedent suffered an injury, end stage cirrhosis of the liver, which arose in the course of his employment at BIW, and she has shown through the credited testimony of Mr. Westlake that the Decedent was exposed to carbon tetrachloride at work. She has also introduced the expert medical opinion of Dr. Pohl that the levels of carbon tetrachloride to which the Decedent was exposed were harmful and that this exposure damaged his liver, resulting in the fatal cirrhosis. I find that this evidence easily satisfies the Claimant's *prima facie* burden.

Once a claimant makes out a *prima facie* case, she is entitled to a presumption that the injury or disease was caused by working conditions and is, therefore, compensable under the LHWCA. *Preston*, 380 F.3d at 605; *Brown*, 194 F.3d at 5. Consequently, the burden shifts the party opposing entitlement to "rebut the presumption with substantial evidence that the condition was not caused or aggravated by his employment." *Bath Iron Works Corp. v. Director, OWCP*, 109 F.3d 53, 56 (1st Cir. 1997). Evidence is "substantial" if it is the kind that a reasonable mind might accept as adequate to support a conclusion. *Richardson v. Perales*, 402 U.S. 389, 401 (1971); *Sprague v. Director, OWCP*, 688 F.2d 862, 865 (1st Cir. 1982). Under the substantial evidence standard, an employer does not have to exclude any possibility of a causal connection to employment, for this would be an impossible burden; it is enough that it produce medical evidence of "reasonable probabilities" of non-causation. *Bath Iron Works Corp. v. Director,*

OWCP, 137 F.3d 673, 675 (1st Cir. 1998). *See also Ortco Contractors, Inc. v. Charpentier*, 332 F.3d 283, 289 (5th Cir. 2003) (rejecting requirement that an employer "rule out" causation or submit "unequivocal" or "specific and comprehensive" evidence to rebut the presumption and reaffirming that "the evidentiary standard for rebutting the § 20(a) presumption is the minimal requirement that an employer submit only 'substantial evidence to contrary.'"), *cert. denied*, 540 U.S. 1056 (2003). The Claimant argues that the Respondents have not met their rebuttal burden because Dr. Harbison relies on "negative evidence" which the Benefits Review Board has held to be insufficient to rebut the LHWCA's presumption. Claimant's Brief at 7. Granted, almost all of Dr. Harbison's report is devoted to a critique of Dr. Pohl's analysis and opinions, and there is almost no discussion of any possible non-occupational causes of the Decedent's liver disease. However, the penultimate paragraph of his report sets forth his opinion that workplace exposure to carbon tetrachloride at BIW could not have caused the Decedent's end stage liver disease and death. EX 1 at 7. Given Dr. Harbison's impressive credentials as an expert in toxicology and his detailed attack on Dr. Pohl's findings, I find that a reasonable mind could accept his opinion as adequate to establish a reasonable probability of non-causation. Accordingly, I conclude that the presumption has been successfully rebutted.

In view of the successful rebuttal, the presumption "falls out" of the case, and the Claimant bears the ultimate burden of establishing causation based on the record as a whole. *Brown*, 194 F.3d at 5. The Claimant can only meet this burden if a preponderance of the evidence establishes the requisite causal connection. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267, 277-280 (1994). While Dr. Harbison raised several questions about Dr. Pohl's opinions and the methodology he employed, I find that Dr. Pohl has provided reasoned answers and adequate support for his opinions. That is, he explained that the Decedent's exposure to carbon tetrachloride at BIW, as described in the credited testimony from Mr. Westlake, was harmful since it was in sufficient concentrations to be detected by humans, a level (at least 10 parts per million) that is nearly 300 times higher than the established permissible exposure level (.03 parts per million). He also provided a reasoned medical explanation of how repeated, non-acute exposures to carbon tetrachloride can cause cirrhosis without contemporaneous clinical manifestation, and he testified that the Decedent's exposure was sufficient to have caused the end stage cirrhosis that was diagnosed in 2002 and proved fatal in 2004. Finally, he ruled out other potential causes of cirrhosis such as hepatitis, alcohol abuse and other sources of chemical poisoning. On the other side of the scale is Dr. Harbison's criticism which, frankly, lost much of its persuasive force when answered by Dr. Pohl. In addition, examination of the medical records, which repeatedly refer to the Decedent's episode of yellow eyes or jaundice after prolonged exposure to carbon tetrachloride, reveals that a critical assumption relied upon by Dr. Harbison, that the Decedent never exhibited contemporaneous symptoms, was plainly wrong. For these reasons, I give greater weight to the medical opinion from Dr. Pohl, and I find that the Claimant has carried her burden of proving by a preponderance of the evidence that the Decedent's end stage liver cirrhosis, disability and death were caused by his occupational exposure to carbon tetrachloride during the course of his employment at BIW.[9] Therefore, the injury is compensable under the LHWCA.

[9] Although I have determined that the evidence is inconclusive as to whether the Decedent's episode of jaundice symptoms occurred while he was working for BIW, this uncertainty is not material. That is, Dr. Pohl's testimony establishes that the Decedent's exposures to carbon tetrachloride at BIW were harmful and sufficient to have caused his end stage cirrhosis even in the absence of contemporaneous clinical symptoms. He also testified that the

E. Compensation and Benefits Due

1. Disability Compensation

Since the Decedent voluntarily retired before his work-related cirrhosis became manifest, his disability compensation is calculated under the permanent partial disability provisions of sections 8(c)(23) and 10(d)(2) of the LHWCA based on the degree of permanent physical impairment rather than economic factors. *See Frawley v. Savannah Shipyard Co.*, 22 BRBS 328, 330 (1989); 20 C.F.R. § 702.602 (2004). The parties have stipulated that the Decedent's disability was 100 percent during the period of August 28, 2003 through March 30, 2004 and that the National Average Weekly Wage of $515.39 is applicable for calculation of any benefits. Accordingly, I find that the Decedent was permanently disabled with a 100 percent impairment from August 28, 2003 through his death on March 30, 2004, and I will award his estate permanent partial disability compensation for this period pursuant to sections 8(c)(23) and 10(d)(2) . The applicable weekly compensation rate of $343.59 is calculated by multiplying two-thirds of the stipulated NAWW ($515.39) by 100 percent. This compensation will be paid by One Beacon as the responsible insurance carrier for BIW.

2. Death and Survivor's Benefits

As a surviving spouse who was married to and living with the Decedent at the time of his work-related death, the Claimant is entitled to death benefits and funeral expenses as provided by section 9 of the LHWCA. *See Griffin v. Bath Iron Works Corp.*, 25 BRBS 26, 29 (1991). The Claimant introduced receipts showing that she paid a total of $4,387.00 for the Decedent's funeral. CX 9. Pursuant to section 9(a) of the LHWCA, which allows for funeral expense reimbursement up to a maximum of $3,000.00, I find that she is entitled to an award of funeral expenses in amount of $3,000.00. I further conclude that the Claimant is entitled to survivor's compensation pursuant to section 9(b) of the LHWCA at the rate of 50 percent of the stipulated NAWW average weekly wage, which equals or $257.70 per week, commencing on March 31, 2004 and continuing until her death or remarriage.

3. Interest

Interest is due on all unpaid compensation including funeral expenses. *Adams v. Newport News Shipbuilding & Dry Dock Co*., 22 BRBS 78, 84 (1989). The appropriate interest rate shall be determined pursuant to 28 U.S.C. § 1961 (2003) as of the filing date of this Decision and Order with the District Director. My order incorporates by reference this statute and provides for its specific administrative application by the District Director. The appropriate rate shall be determined as of the filing date of this Decision and Order with the District Director.

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accumulative effect of the low or intermediate level carbon tetrachloride exposure that the Decedent encountered at BIW over a period of years produced the cirrhosis, rather than any single isolated episode of acute exposure. Therefore, even if it is assumed that the Decedent's jaundice symptoms occurred as a result of an incident of carbon tetrachloride intoxication prior to his BIW employment, the subsequent harmful exposures and liver damage that he sustained while working for BIW make BIW liable for the entire injury under the LHWCA's aggravation doctrine. *See Director, O.W.C.P. v. Bath Iron Works Corp.*, 129 F.3d 45, 50 (1st Cir.1997); *Bath Iron Works Corp. v. Director, O.W.C.P.*, 109 F.3d 53, 55 (1st Cir.1997).

4. Medical Expenses

An employer is liable pursuant to section 7(a) of the LHWCA for those medical expenses reasonably and necessarily incurred as a result of a work-related injury. *Colburn v. General Dynamics Corp.*, 21 BRBS 219, 222 (1988); *Parnell v. Capitol Hill Masonry*, 11 BRBS 532, 539 (1979). As the responsible carrier, One Beacon is liable for all medical expenses reasonably and necessarily incurred by the Decedent and the Claimant in connection with the Decedent's work-related end stage liver cirrhosis. In addition, One Beacon will be ordered to reimburse the Claimant and the Decedent's estate for any payments already made for medical bills reasonably and necessarily incurred in connection with the Decedent's liver disease.

5. Attorney's Fees

Having successfully established her right to compensation and medical benefits through the services of an attorney, the Claimant is entitled to an award of attorney's fees under section 28 of the LHWCA. *See Lebel v. Bath Iron Works*, 544 F.2d 1112, 1113 (1st Cir. 1976). The Claimant's attorneys filed an itemized application on July 13, 2005 for attorney's fees and costs totaling $18,755.74. No objection has been filed to the fee application. Upon review, I find that the fee application complies with the requirements of 20 C.F.R. § 702.132(a) (2004) and that the fees and costs requested are reasonably commensurate with the necessary work done, taking into account the quality of representation, the complexity of the legal issues involved and the amount of benefits awarded. Accordingly, One Beacon will be ordered to pay the Claimant's attorneys a fee in the amount of $18,755.74.

III. Order

Based upon the foregoing Findings of Fact and Conclusions of Law and upon the entire record, the following compensation order is entered:

(1) One Beacon Insurance Company, as the responsible carrier for the Bath Iron Works Corporation, shall pay to the Estate of Phillip L. Nein the permanent partial disability compensation benefits to which Phillip L. Nein was entitled, from August 28, 2003 through his death on March 30, 2004, at the rate of $343.59 per week, plus interest on all past due compensation at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each payment was originally due until paid;

(2) One Beacon Insurance Company shall pay to the Claimant Janice Nein survivor's compensation at the base rate of $257.70 per week, plus the applicable annual adjustments provided in 33 U.S.C. § 910, commencing on March 31, 2004 and continuing until death or remarriage, plus interest on all past due compensation at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each payment was originally due until paid;

(3) One Beacon Insurance Company shall pay to the Claimant Janice Nein funeral expenses in the amount of $3,000.00, plus interest on all such expenses at the Treasury Bill rate

applicable under 28 U.S.C. § 1961 (2003), computed from the date each expense was originally due until paid;

(4) One Beacon Insurance Company shall be responsible for reasonable and necessary medical expenses incurred by the Decedent Phillip L. Nein for treatment of his work-related liver disease, and it shall reimburse the Decedent's estate and the Claimant for any payments already made for medical bills reasonably and necessarily incurred in connection with the Decedent's work-related liver disease;

(5) One Beacon Insurance Company shall pay attorney's fees and costs in the amount of $18,755.74 to the Claimant's attorneys, McTeague, Higbee, Case, Cohen, Whitney & Toker, P.A.; and

(6) All computations of benefits and other calculations which may be provided for in this Order are subject to verification and adjustment by the District Director.

SO ORDERED

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DANIEL F. SUTTON
Administrative Law Judge

Boston, Massachusetts